George Brodie

DYSPNOEA.

The Subject of Dyspnoea covers a large extent of the field of Medicine.

There are but few diseases in some phase of which it does not occur, in many it is the pre-eminent symptom, one which is of very practical importance to the Physician since it frequently affords a valuable clue as to the nature of the disease he has to deal with. Moreover it often lies in his power to remove or alleviate the distress it causes to the patient.

It is my intention in this paper, firstly to deal shortly with the Physiology of Respiration.

Secondly with that portion of its Pathology which is concerned in the mechanism of Dyspnoea rather with regard to the Physics of the Morbid processes than with the pathological changes themselves.

This latter for the sake of system I have attempted to classify under various main heads.

I shall also introduce cases which have come under my notice where they seem to illustrate the case in point.

PHYSIOLOGY.

The Boundary separating the Physiology and Pathology of Respiration is so indefinite that it is sometimes difficult to separate the two - and so I think that a short account of its Physiology will be useful even if it only prevents undue repetition of certain Physiological facts which will have to be referred to later.

Mechanism of Respiration.

The prime object of the act of Pulmonary Respiration is to effect the interchange of the Oxygen in the air and the Co_2 in the blood or in other words to supply oxygen to take the place of the products of combustion in the tissues.

For this purpose it is necessary , not only that the air passages should be pervious, but also that a free supply of blood should be passing through the Lungs, for if the blood stream passing through the Pulmonary Capillaries be diminished or obstructed from any cause, it follows that since the Haemoglobin cannot take up oxygen beyond its limit of saturation, there must be an insufficient aeration of the blood as a whole.

Respiration is brought about mechanically by the expansion of the Thoracic cavity by muscular

effort/

effort aided by Nervous Mechanism, Inspiration alternating with its contraction, effected mainly by the natural elasticity of the Lungs and chest walls, Expiration..

The act of Inspiration diminishes the pressure in the Thoracic Cavity and air rushes in , till the pressure in the alveoli is equal to, or nearly to, the atmosphere pressure - there it ceases.

Then the elastic lungs and chest wall aided to a slight extent by the force of gravity, recoil, and air is driven out of the lungs till again the pressures are equalised.

The Muscles that are brought into play for quiet Inspiration are-
The Diaphragm whose contraction increases the longitudinal axis of the Thoracic Cavity.

The Levatores costarum - The scaline muscles and possibly the Inter-costals which increase the antero posterior and transverse diameters.

The only muscles brought into play during quiet expiration are the Triangularis Sterni and to a slight extent the abdominal muscles - the rest of the act being accomplished as described.

Now the Lungs being enclosed in a perfectly air tight cavity - are not able to contract to their fullest extent, for on the one hand there exists the atmospheric/

atmospheric pressure in the Alveoli and on the other their own Natural Elasticity - so the degree they are able to contract will be the measure of the difference of these two forces. If however the Pleural Cavity is opened so that the atmospheric pressure is equalised on both sides, the lung contracts to its fullest extent, which in the dead body is found to be about one third of their normal size.

Thus there exists in the Lungs a certain quantity of air that is never expelled during quiet respiration this is the Supplemental plus the Residual air amounting to about two thousand three hundred cubic centimetres.

The amount of air breathed in and out during each ordinary respiration is the Tidal air about four hundred cubic centimetres.

By a forced Inspiration an additional quantity of Tidal air can be drawn in, which is called the Complemental air equal to about 2000 c c .

By a forced expiration the Supplemental air can be driven out, leaving the Residual air which is equal to about 600 c c.

The natural rate of Respiration in an adult is about sixteen to twenty per minute but this is altered by various circumstances.

Thus in sleep it is diminished in excitement accelerated.

The//

The will can inhibit the Nervous mechanism and stop breathing for a time.

In Children respiration is naturally more rapid.

The Lungs are not the only organs that effect the elimination of Carbon dioxide. The skin and excretory organs do so to some extent.

But while the total amount of Carbon dioxide discharged from the Lungs in twenty four hours is about eight hundred Grammes, that from the skin, the next important organ in this respect, is only four grammes, it is also known that oxygen can to a very small extent be absorbed by the alimentary canal.

Chemistry of Respiration.

The gases excreted from the Lungs are Carbon dioxide, Ammonia and Hydrogen, the last two only in minute quantities.

Water vapour is also excreted in considerable amount.

How is the interchange of the Oxygen with the other gases brought about?

When air is drawn into the Lungs, it does not come into intimate contact with the walls of the alveoli , the air in there is practically stationary being drawn to and fro, each time the alveolus expands and contracts it consists of the Supplemental and Residual air, this stationary air contains more Co_2/

Co_2 and less Oxygen than the Tidal air.

By the law of diffusion of gases it gives up some of its Co to the Tidal air and takes from it some of its Oxygen tending towards a state of equilibrium on each side of the two gases.

Now with regard to the interchange of the gases of the alveoli and those of the blood the same principal applies. It is necessary that the Partial Pressure of the Co_2 in the Pulmonary artery should be greater than the partial pressure of that in the alveoli in order that diffusion should take place, and conversely the same holds in respect to the oxygen, which has a higher tension in the alveoli and so is able to diffuse into the blood.

That the interchange does take place by diffusion has been proved by an experiment by Wulfberg, who blocked one bronchial branch of a dog by means of a Pfleuger's Catheter, so that respiration and circulation were not generally interfered with, and so was enabled to draw samples of air from the blocked Bronchus.

It was found that the Co_2 increased in amount till its percentage approximated to that of the blood and then became stationary, no further diffusion taking place. The greater quantity of Oxygen taken in goes to oxydise Carbon - but some also/

also goes to oxidise Hydrogen.

Expired air contains about four per cent more carbon dioxide and four to five per cent less oxygen than inspired air. Now if all the oxygen of the oxygen of the inspired air went to oxidise Carbon, the volumes of the gases of the Inspired and that of the Expired air (disregarding for the moment the small quantities excreted otherwise) when measured at the same temperature and pressure should be equal, but the Carbon dioxide is usually in excess in the proportion of about four to five- the difference going to oxydise Hydrogen.

The volume of the Carbon dioxide Expired divided by the volume of the oxygen Inspired is what is called the Respiratory quotient, it is a measure of the quantity of Oxygen used in oxydising Carbon.

It varies according to circumstances, for instance in a Carbohydrate diet which contains enough Oxygen to oxydise all its own Hydrogen it approaches unity.

But in a fat diet which does not contain nearly sufficient Oxygen to oxidise all its Hydrogen, there is less Oxygen available for the purpose of oxydising Carbon so the Respiratory quotient is lowered.

During sleep and in Hybernating animals where metabolism is in a state of inactivity , the Respiratory/

Respiratory quotient is lowered because more Oxygen is inspired then is required for the amount of Carbon burned. But in Fever where combustion in the tissues is active the Respiratory quotient is raised, and the increased rate of respiration then seen may be regarded as a compensatory effort on the part of nature to supply the deficiency in Oxygen and to facilitate the diffusion of the gases.

Internal or Tissue Respiration.

The oxygenation of the tissues is brought about by the arterial blood saturated with oxygen, brought to them in the capillaries.

Such tissue as muscle which may be taken as a type is extremely avid of oxygen. In fact it is impossible to extract free oxygen from muscle under the air pump; it being all greedily taken up and fixed immediately it is brought into contact with it. Thus the Partial Pressure of the Oxygen in the Serum bathing the tissues, is lowered and thus facilitates the diffusion of oxygen from the blood into the Serum bathing the Intercellular spaces.

Production of Carbon dioxide does not appear to be brought about by the direct union of Carbon with Oxygen, and an excised muscle in an air-tight chamber from which all oxygen is excluded can still respond to stimuli and go on contracting for some time/

time and producing Carbon dioxide, therefore the oxygen must be fixed and stored up in the muscle as some Chemical substance, which when broken up yields Carbon dioxide as one of the products.

The greater part of the Carbon dioxide produced is dissolved mechanically in the Serum, part goes into Chemical combination in the Red blood corpuscles. Oxygen on the other hand is chiefly found in loose combination with the Haemoglobin of the Red cells.

Nervous Mechanism.

Although respiration is to a large extent an involuntary act, it is also under the control of will to some extent, or such acts as speaking, coughing and the like would be rendered impossible.

The nerve centre is generally supposed to be situated in the Medulla about the origin of the Vagus Nuclei.

Various experiments that have been made lead to the deduction that the Nervous Mechanism is partly Automatic and partly reflex.

If the Cerebrum be cut off from the Medulla, Respiration continues more or less regularly.

If the Vagi are cut as well, there is an irregular discharge of nervous energy from the centre resulting in Respiratory spasms both of Inspiration and Expiration (Marckwald).

If/

If the Vagi are cut alone respiration is slowed but remains rhythmic in character.

The Respiratory mechanism is affected differently by different kinds of stimulations of the proximal ends of the cut Vagi.

Slight stimulations by Faradism causes quickening of respiration.

Strong stimuli cause arrest in the Inspiratory phase.

Mechanical stimuli such as pressure or stretching the nerve cause slowing of Respiration and finally arrest at Expiration..

The conclusion to be drawn from these and similar facts is that the Medulla is the main motor centre but that it is controlled and regulated by rhythmic nervous impulses coming to it from the Vagi and from the Cerebrum.

It is supposed that the Vagi contain two sets of different fibres and that the termination of the one set in the Lungs when stimulated by its expansion exercise an inhibitory effect on the Inspiration and stimulate Expiration while the other set are stimulated by the contraction of the Lungs and then inhibit Expiration and stimulate Inspiration. (Hering Breuer).

Others infer from the above that the Vagi contain/

contain only one set of fibres and that they are affected differently by different kinds of stimuli.

Afferent impulses also pass to the Respiratory centre from the Superior Laryngeal Nerve which when stimulated excites Expiration and inhibits Inspiration The Glosso-Pharyngeal Nerve on the other hand when stimulated as by the act of swallowing temporarily inhibits Inspiration.

Many other Sensory Nerves such as those of the skin when stimulated excite Inspiration.

Afferent impulses are said by some to pass from the muscles to the centre of Respiration.

It is certain that muscular activity increases the rate of Respiration but this cannot be entirely due to nervous impulses but rather to the resulting increase of waste products in the blood and exhaustion of oxygen.

For if a muscle be entirely severed from its nervous connection and tetanised, the rate of Respiration is increased, almost conclusively proving that the stimulus must come from the action of the blood on the Respiratory centre.

The Respiratory centre is also in relation with the Phrenic and Intercostal Nerves as well as those of extraordinary respiration.

Section of the Phrenic Nerves causes death to take place rapidly - but life can be maintained almost indefinitely without the action of the intercostal/

intercostal muscles.

Motor fibres also run in the Vagi to supply the muscular fibres in the Bronchial tubes, their relation to respiration is not certain.

Automatic Action.

The theory that excess of Carbon dioxide in the blood, alone causes stimulation of the Respiratory centre, though plausible seems hardly to be borne out by experiment.

It is certain that venous blood does stimulate respiration - but there is lack of evidence that excess of Carbon dioxide alone causes dyspnoea provided plenty of Oxygen is present. The evidence from various experiments is somewhat conflicting, but on the whole it goes to prove that both are factors in causing Dyspnoea but that the absence of oxygen rather than excess of Co_2 excites respiration.

The effect of Respiration on the blood pressure.

The increase of size in the Thoracic Cavity during Inspiration produces a decrease of pressure in it as compared with the atmosphere pressure; and the greater the expansion, the more is thus reduced since more of the force is required to overcome the natural elasticity of the Lungs.

Thus it is that the internal air pressure is always somewhat less than the atmospheric pressure, but/

but especially so during Inspiration. The con - sequence of this is to favour Cardiac Diastole since the heart is working in a partial vacuum; more blood is sucked into the right heart; and when it contracts the pressure in the Pulmonary artery is raised, moreover the vascular tension in the Lungs being reduced during Inspiration De Jager. more blood flows through the lungs and into the left chamber of the heart. Therefore at each diastole of the heart that takes place during Inspiration more blood is forced into the Systemic circulation during the following systole and the blood pressure is raised.

During Expiration the Intra - thoracic pressure is raised somewhat, less blood is drawn into the right side of the heart, and the vascular tension in the Lung being raised by the contraction of the inter-pulmonary vessels, less blood is supplied to the left ventricle and the blood pressure is lowered.

The blood pressure and Respiratory curves do not exactly coincide as to their points of Maximum and Minimum intensity.

If a simultaneous tracing is taken of each, the blood pressure curve is seen to rise with the Inspiratory curve, and to continue rising till after the fall commences in the Respiratory curve - then the blood pressure curve falls till just after/

after the Expiratory curve begins to rise.

The above is I believe the theory that is generally adopted at present by Physiologists but there is also another theory which must be mentioned, namely that the rhythmic rise and fall of the blood pressure is due to Nervous impulses projected from the Vaso Motor Centre Synchronously with Inspiration and Expiration.

Asphyxia.

Acute Asphyxia is not often witnessed in the Human subject, such examples as we have in Pulmonary embolism or sudden flooding of the Lungs from bursting of an Aneurism, are rendered impure on account of the disturbance in the Pulmonary circulation which necessarily takes place - and therefore in the circulation as a whole.

When air is suddenly withheld from entering the Lungs the following Phenomena occur.
First - attempts at respiration becoming rapidly more violent, and calling all the extraordinary muscles into play, great muscular excitement, increasing Cyanosis and engorgement of veins.

Secondly a stage in which muscular twitchings increasing to spasm and general convulsions occur.

Thirdly respiration ceases the muscles become flaccid and the pupils dilated.
These phenomena can be interpreted in the following manner/

manner.

First stage the blood becomes venous and stimulates the Respiratory centre.

Second the blood becomes ultra venous and stimulates other motor centres which are normally less easily excited.

Thirdly the nerve centre becomes more and more exhausted as the oxygen is used up till finally they cease to work altogether.

Chronic Asphyxia.

When the organism is gradually deprived of oxygen its deficiency is at first compensated by increased rapidity and depth of Respiration.

But eventually a time comes when the compensation falls to keep up the Haemoglobin to its proper state of saturation with oxygen. There is pallor, muscular weakness and loss of nervous excitability and thus a vicious circle is established. The nerve centres loosing their normal power of stimulating, and the muscles the power of reacting to the stimuli.

Eventually the patients life ebbs away and he sinks gradually from general exhaustion.

As a consequence of impoverished blood supply one sometimes has in this and other conditions a modification of the Respiratory Rhythm called Cheyne Stokes breathing.

The/

The following are the characteristics.
The Inspirations gradually increase in depth till a maximum is attained, then gradually subside till all Respiratory movement ceases, and a period of Apnoea supervenes for a few seconds then an almost imperceptible Inspiration and so on.

It is an indication of extreme exhaustion of the Respiratory centre.

PATHOLOGY OF RESPIRATION.

The morbid processes which may give use to Dyspnoea may be roughly divided into five great classes.

I. Obstruction to air entering the Respiratory passages.

II. Obstruction to the blood passing through the Pulmonary circulation, including Cardiac Dyspnoea.

III. Dyspnoea of Haemic origin, when Haemoglobin the essential element for the transmission of oxygen is wanting.

IV. Dyspnoea of Toxic origin.

V. Dyspnoea of Nervous origin from injury, disease and paralysis of the Nervous Mechanism.

OBSTRUCTIONS IN THE RESPIRATORY PASSAGES.

The nose and Naso Pharynx are common enough sites for obstruction but since the mouth remains available for use, they do not usually cause dyspnoea.

Obstruction in the pharynx occasionally happens generally in relation to some localised septic condition such as Retro - Pharyngeal abscess, or with inflammation/

inflammation of Oedema of the Pharynx which may supervene during Scarlet Fever or Diphtheria.
Only a short time since I was called to see a child of eighteen months who had Scarlet Fever, and whose submaxillary glands were so enlarged and pharynx so oedematous and inflammed, that it was almost at the point of suffocation. There was scarcely any entry of air into the chest - the intercostal spaces and lower ribs were retracted at each Inspiration and the child was Cyanosed and would no doubt have died if Tracheotomy had not been performed a little later.

OBSTRUCTION IN THE LARYNX.

The narrow slit of the glottis is especially prone to become obstructed.

When this is the case the character of the breathing is characterised by the following features, which generally speaking may be said to apply to all obstructions in the upper air passes above the Bifurcation of the Bronchi.

In the first place there is, generally speaking more or less stridor most commonly with Inspiration, but also occasionally, with Expiration..
Secondly and this is an important point, the rate of Respiration is invariably lowered, and is much deeper/

deeper than normal, according to the amount of the obstruction.

If the obstruction be to the entrance of air as may be seen in the case of some peduncilated Tumour situated above the glottis and acting like a valve, thus Inspiration will be slow and laborious and the extraordinary muscles of Inspiration brought into play.

Auscultation reveals a prolonged and weak Inspiratory sound, coupled with much muscular rumbling. The intercostal spaces are sucked in at each Inspiration the lower ribs are pulled inwards by the action of the Diaphragm - while Expiration is normal . In short there exists Inspiratory Dyspnoea. But if the obstruction to the exit of air, as by some body acting like a valve below the glottic opening then the Dyspnoea is Expiratory and such extraordinary muscles of exspiration as the abdominal muscles will be brought into play.

If the opening is stenosed then both Inspiratory and Expiratory Dyspnoea are present but the former predominates and is the commoner.

It has already been mentioned that contraction and expansion of the lungs stimulates Inspiration and Expiration. Hering Breuer's law, if this is so then it necessarily follows that any impediment to the entrance of air or its egress, must result in slowing /

slowing of the Respiration.

This and the deepening of Respiration is an attempt on the part of the muscles of Respiration to reduce the pressure in the Thoracic Cavity to the fullest extent possible, in order that the air may be more forcibly and rapidly drawn through the narrow opening, and it might be compared with the slow and powerful contractions of the left ventricle in a case of well compensated Aortic Stenosis.

In fact the extraordinary muscles of Respiration may be regarded as a reserve force for the purpose of Pulmonary compensation - and when they fail as they too often do in the case of young children with Laryngeal obstruction the result is just as serious as failure of the Cardiac compensation.

Kohler has shewn in a series of experiments on animals, whose Trachea he compressed that at each inspiration and expiration a considerably greater quantity of air was drawn into the lungs than was the case with an ordinary inspiration ; so much more, that in a given time the amount of air was very nearly equal to the normal - this shews how it is that many persons suffering from a considerable degree of Laryngeal stenosis may live for years in good health, provided what I have called Pulmonary compensation remains incompared.

The/

The causes of Laryngeal obstruction may be divided into Inflammatory and Non Inflammatory. Simple acute Laryngitis may develop acute oedema of the Glosso Epiglottic folds with more or less sudden blocking of the Larynx. Occasionally the oedematous material becomes infected with pyogenic organisms and an abscess forms Acute oedema may develop in the course of fever such as Scarlet fever.

In typhoid fever the Larynx is often the seat of inflammatory infiltration and later on Ulceration may develop. Healing of the ulcers leads to cicatricial contractions and stenosis of the glottis ; acute oedema is sometimes seen and Perichondritis may destroy and distort the glottic opening.

Small Pox is often associated with Laryngitis, with a particular eruption in the Larynx - leading to ulceration and frequently to Perichondritis with marked destruction of cartilage and subsequent great distortion and contraction of the Larynx.

Laryngeal Erysipelas is also described, when it occurs it is very fatal death being due to suffocation generally.

A case of chicken pox is described by Halle when death was due to acute oedema of the Larynx.

Diphtheritic Laryngitis /

Diphtheritic Laryngitis is perhaps the commonest cause of laryngeal obstruction especially in children - and it is particularly fatal on account of the intoxication and muscular weakness which is associated with it, not only is the Larynx swollen and oedematous, but there is also the membrane which may grow over and completely block the orifice.

Tubercular Laryngitis is a common cause of of laryngeal stenosis - chiefly on account of the ulceration and destruction which accompanies it.

Syphilis in the tertiary stage produces marked and rapid destruction of tissue and cartilage with stenosis after healing, and frequently with web like hands stretching across the larynx.

Gummata may develop in the vocal cords and impede respiration.

Inflammatory reaction following injury may produce oedema, often seen in cut throat.

Non-Inflammatory causes.

Oedema of the larynx may develop during the course of renal or cardiac disease.

From pressure of Mediastinal tumours on the large veins.

In the course of Angio Neurotic Oedema as in the following case.

A.N./

A.N. boy aged 15, admitted with injured leg. Family History good, no similar history in rest of family. General health excellent.

While in hospital he occasionally developed swelling on various parts of his body, which appeared suddenly in an hour or two, and disappeared as suddenly, they sometimes reached the size of about half a bantams egg - and were accompanied by very little inconvenience or pain except slight colic. One day, however he complained of swelling in his throat and rapidly became very Dyspnoeic - but it passed quite away in about an hour and a half.

The case was diagnosed as one of Angio neurotic oedema and I think there is no doubt that the dyspnoea was due to a swelling in the larynx of the same nature as the other swellings.

Oedema of the larynx may be caused by taking Potassium Iodide Fournier and Rosenberg.

Paralysis or Spasm of the muscles which open and close the glottis are often a cause of impediment to the entrance of air.

Paralysis of the abductors is the commonest form.

The cause may be some gross organic lesion of the Cerebro Spinal system and as Locomotor Ataxy, Bulbar paralysis or Amyotrophic lateral Sclerosis.

Most/

Most commonly it is due to inter-thoracic growth and most frequently of these to Aneurysm of the arch of the Aorta for the reason that the left recurrent hooks round the arch of the Aorta so is liable to become involved with it when it is diseased.

The effect is paralysis of the abductors of the cords on the left side.

The recurrent laryngeal also supplies the adductors but it is found and has been stated as a law by Senson, that in any form ofgross Lesion of the recurrent laryngeals the abductor fibres are the first to suffer.

Consequently when the left recurrent is implicated in such a manner it is usual to find the left vocal cord immovable and drawn towards the middle line by the unopposed action of the adductors.

Now single left sided abductor paralysis is not capable of producing sufficient stenosis to cause Dyspnoea.

How then can one explain the sudden and violent attacks of Dyspnoea that these patients suffer from at times, while at others they may be free from all such symptoms.

In the first place it may be due to direct pressure of the Aneurism on the Trachea, but there are/

are many cases on record of this kind of Spasmodic Dyspnoea when it has been proved that no such pressure ever existed.

Secondly it may be due to bilateral paralysis of the abductor fibres from implication of both recurrent laryngeal nerves.

This always produces narrowing of the glottis sufficient to produce Dyspnoea but it does not explain the isolated attacks.

Mott gives the probable solution when he suggests that the same lesion which paralyses the abductor fibres sets up a state of irritation in the adductor nerve fibres so causing a spasm of the adductors of the glottis.

Johnson has suggested that long continued irritation of the trunk of the Vagus may so affect the Vagus Centre as to cause bilateral spasm of the glottis adductors, combining a state of abductor paralysis and adductor spasm on the left side with adductor spasm on the right side.

Thirdly it may be caused by contraction of the Bronchial muscles - from irritation of the trunk of the Vagus acting on those fibres which supply the circular muscles of the Bronchi.

The following case may be an example

A. B. seaman aged 56.

Admitted/

Admitted complaining of pain in the chest.
There was a distinct history of Syphilis and Alcohol.
Examination of the chest revealed slight dullness over upper part of the Sternum - and there was a systolic murmur in the same region.

The left radial pulse was delayed and the left local cord paralysed.aneurism was diagnosed.

Later on the patient became subject to violent attacks of Dyspnoea which came on suddenly and were Asthmatic in character, the patient had to sit up and grasp the support with his hands.

There was no obstruction to the entry of air through the larynx, yet entry of air into the chest was very deficient and exspiration was very prolonged laboured and wheezing - during an attack he became very cyanosed.

Nitrite of Amyl relieved the spasms as a rule.

The P. M. revealed a Saccular Aneurysm of the arch of the Aorta which involved the left recurrent and the trunk of the vagus - there was no evidence of pressure in the trachea or bronchi.

Complete Recurrent Paralysis.

All the muscles of the glottis being paralysed the cords lie in the Cadaveric position with a narrow oval chink between.

There is usually Dyspnoea and Stridor. The Dyspnoea as a rule is of the Inspiratory order owing to/

to the suction of the cords together from the Inspiratory rarefaction of the air below.

Inspiratory Dyspnoea is also seen in Bilateral paralysis of the Postice.

This is a rare affection and when present usually depends on paralysis of the special branches of the inferior Laryngeal which supply them . The consequence of this paralysis is to secure the abnormal effect of closing instead of opening the glottic aperture during inspiration - so dyspnoea during Inspiration is usually very marked.

Spasm of the Adductors.

Laryngismus Stridulus is an affection not very uncommon in young children who are rickety and ill nourished.

It has been attributed by some to a reflex stimulation of the Respiratory centre, by others to cerebral irritation - Horsley.

The attacks are characterised, by stridor and Inspiratory Dyspnoea followed by a short period of Apnoea and ending with a deep inspiration.

Another condition also seen in young children and resembling the latter is the infantile respiratory spasms of John Johnson here the stridor is more or less continuous and the Cyanosis not so marked or absent, while there is very little actual Dyspnoea.

The/

The Epiglottis is found to be folded over on itself and the Aryteno Epiglottic folds approximated, "Lack." Hyperplasia of the Thymus gland is also said to be present in some cases, Avellis.

Hysterical patients are apt to have attacks of laryngeal spasm .

It also constitutes crisis of Locomotor Ataxy.

The type of Respiration is the same in all namely Inspiratory Dyspnoea and Stridor.

Obstruction in the Trachea.

With regard to obstruction in the Trachea little need be added, The type of respiration is very similar.

Acute Inflammation is not so liable to cause obstruction here on account of the size of the Lumen which would require to be filled up by inflammatory exudation, and for the same reason only large tumours inside the trachea would be likely to cause Dyspnoea.

Granulations from Tracheotomy wounds may fill the Trachea.

Tumours and foreign bodies in the oesophagus may ulcerate into and bulge in the posterior wall.

External pressure from aneurysm and other inter - thoracic growths and Goitre among growth in the neck are the most frequent causes of Stenosis of the Trachea.

In the case of Goitre the Trachea is often narrowed, it may be also distorted and bent out of shape./

shape.

One of the results of pressure on it is to cause a softening of the cartilaginous rings which may evidently have undergone some absorption or else show no signs by inspection, yet have lost their elasticity and supporting power, so that when the head is bent the lumen of the Trachea becomes kinked when in the same way as an indiarubber tube is bent.

This has been known to cause death by suffocation.

Goitre and other tumours in the neck may also cause dyspnoea by pressure in the Recurrent Laryngeals.

Aneurysm of the Aorta may give rise to similar pressure effects on the Trachea and it may be difficult to distinguish between the symptoms due to this and the spasmodic dyspnoea before alluded to.

They may be differentiated so far as the Dyspnoea is concerned by the intermittent character of the spasmodic attacks in the former.
By the absence of laryngeal symptoms in the latter, though these may and often do accompany it.

By the more constant character of that due to direct pressure, and finally it might be added by the fact that in the spasmodic form the Dyspnoea is Expiratory as well as Inspiratory.

The Dyspnoea due to direct pressures on the Trachea/

Trachea is usually Inspiratory since the cartilaginous rings having become so softened that they loose their resilience, the walls of the trachea tend to become sucked together by the inspiratory rare - faction of the air below the obstructure whereas the exspiratory spasm is due in some instances I believe to altogether another influence namely that of the vagi acting on the muscular fibres of the small Bronchi.

Obstruction in the Bronchi and air vesicles.

While it was possible in the case of the upper air passages to generalise somewhat on the character of the Dyspnoea produced by obstruction, this is not so in the case of the Lungs themselves, on account of the more varied and complicated factors involved.

The trachea was simply a passage for the ingress of air, and it was only necessary for it to be patent in order to discharge this function.

The Bronchi and air vesicles are more complicated in their relation to Dyspnoea. Moreover the factor of the Pulmonary Circulation is inseparably involved in most of the Pulmonary lesions.

In so far as the function of the Bronchial tubes is concerned, it differs in no way from that of the trachea - but their situation renders them less liable to the effects of external pressure than is the/

the case with the latter - and it is obvious that the effect of obstruction even of a large Bronchus is not nearly so severe as is the case of the trachea, since there will always remain a large amount of aerating surface available through other tubes.

Pressure may be exercised on the Extra-pulmonary Bronchi by enlarged Bronchial glands- Aneurysm of the Aorta which chiefly affect the left Bronchus and other Interthoracic tumours.

Inside the lungs the Bronchi are less liable to external pressure, but examples may be found in Peribronchial Tubercular deposits and new growths generally metastatic invading the lung. These last however rarely cause dyspnoea and it is remarkable to what an extent a lung may become infiltrated with cancer without causing any distress of breathing - I recollect such a case in the Medical wards at Edinburgh Royal Infirmary when he had been admitted some months after having his leg amputated for Sarcoma. During life there was no respiratory trouble except slight cough - and the Physical signs only ammounted to patches of moderate dullness, and a few moist râles - yet at the Post Mortem his lungs were literally riddled with masses of sarcoma from the size of a pea to a hen's egg in size.

It may be stated as a general law that the more rapid/

rapid the onset of the obstruction whether it is in the Bronchi or parenchyma of the lung, the more severe will be the effects so far as Dyspnoea is concerned.

The effect of complete obstruction of a Bronchus is of course to put out of action the alveolar territory belonging to it.

The oxygen and the other gases in the isolated alveoli gradually became absorbed and that part of the lung became completely atelectatic. The oxygen disappears first the carbonic acid goes next and the Nitrogen last owing to its low co-efficient absorbtion, Lichtheim -the diffusion of the gases being kept up by the elasticity of the Lung causing it to contract and so keeping up the partial pressure of the contained gases.

The effect of partial obstruction of a large Bronchus is rather on the contrary to produce Emphysema in its alveolar territory, its mechanism being the following.

That amount of air that does find access to the alveoli, has more facility in entering than in leaving the alveoli, for the reason, according to Biermer, that the Bronchi are liable to have their lumen still further reduced by the compression of the lung during its contraction in the act of expiration.

Thus the alveoli are kept in a state of Inspiratory/

Inspiratory expansion, this tells at last on their elasticity and they become overdistended, the septa bacomes thin and may rupture.

Of course if cough is present it will hasten the process on account of the increased intrapulmonary pressure it induces.

Acute Bronchitis.

The Intra pulmonary Bronchi being comparatively individually small are obviously more liable to have their lumen narrowed by inflammatory swelling or spasm, and to become choked with mucus and other products of Bronchial Catarrh. In a case of ordinary acute Bronchitis of the larger Bronchi, the tendency is towards an incomplete blocking of the tubes, with the effect as first stated, that there is not only obstruction to the entry of air but also more especially to its exit, thus a certain amount air becomes imprisoned in the alveoli beyond the affected Bronchi, preventing their normal contraction and entailing abnormal efforts to empty them. There is more or less Cyanosis. The inspirations are rapid, expiration prolonged in proportion. The Thorax is inclined to be expanded and the ribs do not move much on inspiration, but there is little or no sucking in of the Intercostal spaces.

The chest is resonant all over.

These are bubbling rales of various sizes and bronchi among which the vesicular murmur is audible, with/

with prolonged expiration.

In capillary Bronchitis on the other hand and in most cases of Broncho Pneumonia the tendency is towards a complete blocking of the minute Bronchioles with collapse or Hepatization of the alveoli.

There is deficient entry of air as can be proved by the almost inaudible vesicular murmur and by the fact that the chest walls scarcely expand at all during Inspiration. The percussion note may be somewhat less resonant than in the previous case. Râles and Ronchi are not a marked feature, for their presence implies that air is passing a constriction or forcing its way through fluid - such as are present are of a very fine character.

The Respirations are very rapid, with Inspiratory indrawing of the Intercostal spaces, lower ribs and epigastric region.

Later the muscles fail and the respirations become more shallow and more rapid. The child lies exhausted and helpless, bathed in sweat, its face of a livid pallor, pulse rapid, feeble and irregular, extremities cold and cyanosed.

It is in these cases that Oxygen is of the greatest value, but when the lungs are extensively involved the child almost invariably dies in spite of Strychnine Digitatis, Brandy or any other stimulant/

stimulant.

The explanation of the relation of the Intercostal spaces and lower ribs just referred to is as follows The lungs not being able to expand on Inspiration while the Thoracic cavity is increased in capacity, the internal atmospheric pressure becomes greater than the air pressure in the thorax with the result that the soft intercostal spaces are pushed inwards. With regard to the indrawing of the lower ribs, the traction of the diaphragm which is attached to them is chiefly responsible.

Bronchial Asthma.

It might be said, not incorrectly that the Dyspnoea that occurs in Bronchial Asthma has some similarity as regards its mechanism to that seen in Bronchitis.

Of course the Inflammatory swelling and secretion is absent - but its place is taken by a Hyperaemic swelling of the mucosa of Vaso Motor origin, or as some say by a spasm of the circular muscles of the small Bronchi.

The spasm is reflex in origin and may be set up by various kinds of local irritation either in the lungs themselves by breathing cold air, irritating particles and so on - or the exciting cause may be a disordered stomach - or any organ to which fibres of /

of the Vagus are distributed - and to refer to page 34 , I believe that the paroxysmal attacks of dyspnoea seen in Aneurysm may be produced by an irritation of the vagus acting in the Pulmonary Vaso Motor system or on the circular muscles of the Bronchi.

The effect of the spasm or Hyperaemia is to imprison air in the alveoli, and keep the chest in a state of Inspiratory expansion necessitating powerful action of the extraordinary muscles of Inspiration to draw sufficient fresh supply of air into the lungs and still more powerful expiratory efforts to squeeze the air out of the overdistended air vesicles.

When the attack is over respiration gradually resumes its ordinary type.

Before leaving this subject it must be mentioned that spasm of the diaphragm is regarded as a cause of Asthma - during the spasm the cavity of the thorax is increased and further inspirations limited to the action of the ribs, consequently the Inspirations are short, while the expirations are laboured and prolonged.

Bronchiectasis.

Although Dyspnoea is often associated with Bronchiectasis, this cannot always we said to be the/

the cause of the Dyspnoea but rather the effect of the lesion producing the Bronchiectasis.

That there is a form of Bronchiectasis which may produce dyspnoea however, seems to be evident from the following case, which I should say had originally been such a case described by Dr. Sharkey as Acute Bronchiolectasis.

As it was an interesting case it may be worth while to describe it at some length. The patient was a little girl aged seven. She had had a cough more or less since she was two years of age, when she had an attack of Bronchitis after measles. The cough did not leave her, and occasionally (about once a week) she vomited large quantities of muco-purulent material, very foul smelling. Just before each attack of vomiting she always had a short period during which her breathing was very distressed. In appearance she was cyanosed. Finger tips markedly clubbed. Breath very foul and she generally shewed the effects of ill nutrition.

Physical examination shewed nothing particularly noticeable about the shape of the chest. The Physical signs were those of medium sized cavity formation in the lower lobes on both sides, but especially the left where there was dullness to percussion and Bronchial breathing with coarse bubbling râles.

The/

The disgnosis of Bronchiectasis was made and she was in Hospital for many weeks but she did not improve under treatment and eventually died.

A post mortem was performed and the appearance of the lungs was as follows. There were a few slight adhesions between the Pleurae.

Upper lobes of both lungs crepitant and shewed some Emphysema, otherwise normal.
Lower lobes, the central portions near the roots shewed merely Interstitial Pneumonia. The peripheral portions were riddled with small dilated Bronchi, which gradually expanded into branching lobulated cavities, from the size of a goose quill to three quarters of an inch in diameter, these extended to the surface of the Pleura which they bulged out, they contained a considerable quantity of muco pus, and their walls were smooth, thick and lined by pavement epithelium .

That portion of the lung was quite non-crepitant flabby and leathery to the touch and sank in water.

There was no bullous Emphysema present and it was impossible to make out any lung tissue near the surface.

So marked was this that it was difficult to believe that certain terminal portions of the cavities were not altered Alveolar portions of the lungs.

The possibility of such a thing occurring might/

might be explained as follows.
The original Bronchitis had produced a blocking of the small Bronchi near the surface, and consequently collapse of the lung in their alveolar territory.

Transudation of serum followed into the collapsed alveoli filling them, and their walls might easily become ruptured and form large spaces. When the contained fluid becomes purulent inflammatory thickening of the walls of the spaces might be produced.

Subsequent cicatrical changes would help to produce further Bronchiectasis.

However the point I wish to lay special stress upon is the relation of the vomiting , if I may call it so and the Dyspnoea. It was difficult to determine exactly whether the purulent matter discharged came from the lungs or whether it was simply matter which had been coughed up swallowed and afterwards rejected by the stomach.

If it came from the lungs then it may be assumed that it came from reflex contractions of the muscles in the walls of the cavities.

Examination of the rejected matter shewed no signs of stomach contents.

The marked Dyspnoea before the vomiting might it is true be due to a reflex effect from irritation of the stomach. But taking one consideration with another and remembering that the Dyspnoea occurred just/

just before the rejection of the muco purulent material and at no other time; it seems reasonable to suppose that it (the Dyspnoea) was due to a reflex contraction of the small bronchi, induced by the overdistention and irritation of their walls, the effect being at the same time or a little after to express their contents.

This is a point of some interest as regards whether there is destruction of the Bronchial muscles in this disease, as alleged by Bamburger - while it supports the view of Cohnheim that the irritation of the retained secretion may produce contraction of the Bronchial muscles.

Emphysema.

Of the varieties of Emphysema that called by Jenner the large lunged variety, is the one which is of the greatest importance for the present consideration.

As a rule it implies a general cause which may exert its influence on a large extent of both lungs.

Compensatory Emphysema on the other hand is merely a condition called into existance by some local contracting lesion of the lung and its extent will be proportionate to this.

The compensation does not necessarily apply to the/

to the regulations of the respiratory process, but merely to the adjustment of the bulk of the lung to the capacity of the thoracic cavity.

The effects of Emphysema are due to three principal causes.

1. Increase in bulk of the lungs.
2. Diminished total alveolar surface.
3. Secondary Cardiac changes.

The increase in bulk of th lungs implies overdistention of the normal capacity of the thoracic cavity.

The lungs are kept ina state of Inspiratory expansion not for the reason that air is imprisoned in the alveoli, as we saw in Bronchitis but because the lungs have lost their elasticity and the atmospheric pressure has it all its own way, and tends to keep the air spaces filled, so the capacity of the chest can only be to a small extent augmented even by forced inspiration.

The expansion of the Thorax is regarded by Stokes as the result of overaction of the Inspiratory muscles, which have lost the opposing action of the elastic lung tissue and which therefore keep the chest walls in a state of Inspiratory expansion. If one were to criticise this it might be said that one would expect the Inspiratory muscles in such a case would tend to atrophy as the result of loss of/

of opposing action, but as a matter of fact they are markedly hypertrophied.

Dr. Harry Campbell in a recent paper states that the expansion of the chest is a position involuntarily assumed by the patient to lower the vascular tension in the lungs as much as possible and favour circulation of blood through them, and the Inspiratory muscles become abnormally developed as a result of this constant activity in promoting this state of affairs. At all events it is only by forced expiration that the Thoracic cavity can be diminished even to the small extent usually possible on account of this loss of elasticity and because the Diaphragm can't assume its normal dome like shape without help of the abdominal muscles. Nor is this all - Respiration is slower than usual owing to the delay which takes place in the discharge of the stimulus which should occur at the end of Normal expiration. Hering Breuer. Possibly also if this theory is correct there may be an abnormally powerful stimulation of the special fibres of the Vagus from the Inspiratory expansion of the lungs which may produce a reflex contraction of the Bronchial muscles as well.

This might explain the severe acute spasms of Dyspnoea which patients with Emphysema are liable to.

But it might be said if this is so that the patient should never be free from such attacks.

A/

A reasonable answer to this would be, that the cells discharging this superfluous energy would soon become exhausted and rendered incapable of discharging more than their usual quantity, till a state of rest again restored their vitality.

2. Diminished total alveolar surface.

This implies not only less available surface for exposure to air, but also even more important, a diminished capillary area as well - for not only is the capillary area lessened but it may be in parts completely destroyed so that the pressure of air in the spaces avails nothing since no blood can gain access to it.

This obstruction to the flow of blood through the lungs throws great strain on the right ventricle which must hypertrophy in order to overcome the resistance. Later on compensation is apt to fail and the patient may die of Cardiac failure.

This will be again referred to under the second head.

If the Respiration of a patient suffering from this disease is watched, it will be seen that, the number of Respirations per minute are less than usual. That Expiration is especially prolonged and attended by great effort.

While the chest walls remain expanded and rigid all/

all the time, the abdominal muscles apparently do most of the work and shewing probably development disproportionate to the rest of the muscles. The Sterno Mastoids and the muscles of forced Inspiration are also greatly developed but are unable to produce much apparent effect. Such patients are frequently the subjects of chronic Bronchitis the Catarrh and secretion caused by it also tend to aggravate the Respiratory embarrassment.

In Senile Emphysema.

Here the effect produced on the Respiration is due chiefly to diminished alveolar surface.

The lungs are small and do not bulge out the chest wall - in fact the chest walls are on the contrary retracted as in Exspiration and their defective mobility is due to want of muscular power in the first instance, rigidity following as a secondary effect.

This condition does not produce Dyspnoea since it occurs in old people whose metabolism is inactive, the supply of blood to the lungs accommodates itself to the small demand of oxygen on the part of the tissues.

Consequently there is less strain on the right side of the heart.

In Pneumonia./

In Pneumonia.

The Dyspnoea in this disease is chiefly due to obstructions to the entrance of air into the alveoli. To a subsidiary extent it is due also to congestion and stasis of the blood in the Pulmonary capillaries with enlargement of the right heart.

I say to a subsidiary extent, not because it does not constitute a very important part of the disease, but since the alveoli are blocked it is a matter of small consequence, for the purpose of oxygenising the blood, whether it circulates through them or not.

The degree of Dyspnoea will of course depend on the extent of lung involved. One often meets with a case however where the rapidity of breathing is not of all proportionate to the extent of the consolidation, which may be insufficient to justify one in ascribing the dyspnoea to secondary cardiac changes.

Nor could it be due to loss of aerating surface, since plenty of healthy lung may remain to carry on respiration.

The Dyspnoea may be accounted for in several ways. First it may be and probably is, to a large extent due to the fever present, the tissues requiring more oxygen than usual owing to the rapid combustion that is going on.

That this is so is evident if a patient suffering/

suffering from Pneumonia is examined before and after the crisis.

Before: the respirations are rapid even though he has plenty of healthy lung. But after the crisis his respirations fall almost as quickly as the temperature from thirty or forty per minute to the normal number and are quite easy.

Secondly the rapidity may be due to the presence of Pleurisy, the pain causing the patient to take shallow respirations, and so, in order that he may take in sufficient oxygen the number of respirations have to be increased.

Thirdly it may be due to the rapidity of the onset of the consolidation - the respiratory mechanism not having time to recover its balance, so to speak. Shallowness and rapidity are the characteristics of the respiration , the chest wall is always expanded more than usual as can be proved by measurement during the disease and after - this is for the purpose of favouring circulation and partly to obviate pain by lessening the extent of movement.

In Phthisis and acute Tuberculosis.

Nothing much more remains to be added in regard to these - except that the Dyspnoea is less in the case of the more chronic forms of Phthisis, than in the case of Pneumonia and acute Tuberculosis for reasons/

reasons that are evident from the foregoing.

In Phthisis sudden Dyspnoea may be produced by the bursting of an aneurysm in a cavity and flooding part of the lung with blood, also by occurrance of a Pneumo-thorax of which more later

In Oedema of the Lung.

Dyspnoea in Oedema of the lung is always serious, since the exciting cause is generally one that produces general oedema - and is due to some grave disorder such as Bright's disease or Cardiac failure.

Its effect is to choke up and block the alveoli, but not to the same extent as a croupous infiltration. Moreover, both lungs are equally likely to become involved, and that rapidly in some cases.

The type of Dyspnoea is not of any special type, the respiration being merely accellerated.

Effects on Respiration of external pressure on the Lung.

Since the Thoracic cavity is a closed one it follows that any encroachment on its space must be at the exposure of the lung chiefly- since that organ occupies the largest area and is the least resistent of the Thoracic organs.

The effect of pressure on it is to prevent its proper expansion and consequently the entrance of air/

air into the alveoli by the part pressed on. The result is absorbtion of air in the alveoli and Pulmonary collapse in that position to an extent sufficient to adapt the size of the lung to the altered Thoracic capacity.

Such causes of pressure may be Pleurisy with effusion, Hydrothorax Pyo-thorax - Pneumothorax, large abdominal tumours or ascites preventing the Diaphragm from contracting. Intestinal tumours - Pericardial effusions, Deformity of the spine such as Scoliosis.

Since the effects on respiration are generally speaking similar, description of one or two varieties will suffice.

Pleurisy with effusion.

The pressure of fluid in the Pleural cavity will be attended by more or less Dyspnoea, according to its amount. Rapidity of accummulation and ability of the Respiratory mechanism to regulate its balance.

Part of the balancing mechanism is due to compensatory hypertrophy in the healthy lung, by which its volume is augmented and its elasticity increased.

Part to compensatory balance in the heart, and part is due to the fact that the portion of the/

the lung pressed on becomes atelectatic. For if the volume of the lung remained as before, then with a quantity of fluid in the cavity as well, it can only expand if the Thoracic cavity is increased by forced inspiration. But if the process takes place sufficiently slowly to allow effusion and collapse to take place side by side - then so long as a reasonable quantity of lung tissue remains, Dyspnoea will be absent - or only manifests itself in slight accelleration - the respiratory movements on the affected side being also more shallow when compared with the healthy side, on account of the diminished volume of the lung.

On the other hand if the effusion takes place so rapidly that collapse cannot or does not keep pace with it, then Dyspnoea will occur even with comparatively small quantity of fluid.

In Pneumothorax.

The same general principal applies here as in the last - but it differs in that the intrapleural tension is equal all over the affected side- so if there are no Pleuritic adhesions and the intrapleural pressure is equal to that of the atmosphere, the whole lung will collapse to the same extent, on account of its elasticity asserting itself.

In the case of a closed Pneumothorax, Respiration will not be much interferred with, so long as the Intrapleural tension, by which I mean the atmospheric force which normally keeps the pleural surfaces in contact/

contact; is greater than the elasticity of the lungs.

The valvular Pneumo thorax has the most serious consequences. Air is able to rush into the Pleural cavity during Inspiration when the Interpleural Tension is lower owing to the expansion of the Lungs, but can't escape during Expiration when the interpleural tension is greater.

The consequence is that the air pressure in the Pleurae gradually rises, till it equals that of the atmosphere. It can never become more, except in the case of forced Inspiration, for when the cavity of the Thorax is reduced again, the air not being able to escape, owing to the external force keeping the valve closed, it is imprisoned in a space less than that which it entered at the Atmosphere pressure.

Not only are the effects of Pneumo Thorax, as well as fluid exudations, to be found on the affected side, but also on the previously sound side - because the atmospheric or other pressure forces the heart and Mediastinum over to the opposite side, so diminishing the capacity of the opposite side of the Thorax, and hindering the expansion of its lung.

Hence it is not to be wondered at that a Pneumo thorax, occurring as it is apt to do suddenly - is attended by such severe respiratory symptoms. At the/

the first onset the Dyspnoea is as a rule very marked especially during Inspiration, indicating attempts on the part of the Inspiratory mechanism to overcome the unaccustomed resistance to the expansion of the lungs. And the respiration though attended by more effort are rapid and shallow for the reasons that the chest is already so distended that further expansion is impossible.

The general type will be modified however by various circumstances.

The most severe effects are seen when the lungs are previously healthy. But if as is generally the case the elasticity of the lungs is impaired by chronic inflammation, or Pleuritic adhesions which may limit the process and prevent them from contracting to the same extent, the effect produced is less on both sides of the chest and general result in the respiration of the patient less obvious.

Obstruction to the flow of Blood through the Lungs including Cardiac Dyspnoea.

In no group of disorders is Dyspnoea so generally seen as in disorders of the organs of the circulatory system - and as has already been mentioned, the respiratory difficulty in many diseases of the Lungs is in no small part due to existing embarrassment of the heart.

Assuming that there is no obstruction to the entry /

entry of air, it is also necessary for the proper interchange of gases that the blood should flow freely through the lungs.

Various hindrances may exist to this. The heart itself may be at fault as in the case of mitral incompetence and stenosis fatty heart, or the obstruction may exist in the lungs as in Emphysema. Compression of the lung by gas or fluids, atelectasis Croupous consolidation - Pulmonary embolism . But whether the obstruction exists in the lungs or in the heart outside the lungs as in Mitral disease or whether the Pulmonary circulation is retarded by deficient vis a tergo as in tricuspid incompetence, the effect in one respect is the same, unless compensation is perfect, namely that less than the normal amount blood passes through the lungs in the unit of time.

Significance of Dyspnoea in Cardiac disturbance.

Considerable deviation from the normal may exist without exciting Dyspnoea as long as the muscular substance of the heart retains the power of contraction sufficient to overcome the resistance or in other words if compensation exists- all is well. But assuming that there is no obstruction to air entry- Dyspnoea when it does exist seems to serve no useful purpose as Filehne has said.

For /

For when after compensation has failed or for any other reason the blood circulates slowly and with difficulty through the Pulmonary capillaries, it has more than ample time to become saturated with oxygen, any further supplies of air would be wasted on it, therefore why the Dyspnoea.
The cause can be explained by the fact that the Medulla is poorly supplied with arterial blood, and owing to the general backward pressure, the venous blood is dammed up in it, so there exists both the factors of deficiency of oxygen and excess of Carbon Dioxide to excite the Respiratory centre.
Now with regard to the end the Dyspnoea serves.
It is not entirely for the purpose of augmenting the intake of air, but rather by the contractions and expansions of the Thoracic cavity to facilitate the circulation of the blood though the lungs. Cohnheim.

In cases when the obstruction exists to the outflow of blood from the lungs as in mitral disease, where the blood is dammed back in the Pulmonary veins by the regurgitant stream, the Intrapulmonary blood pressure is raised, thus there is more work on the Right ventricle, but so long as this is strong enough to overcome the resistance in front sufficiently to allow the normal quantities of blood to pass through the lungs in the unit of time, Respiration will not be interfered with.

But/

But directly compensation fails or a more powerful call is made on the heart in consequence of some strain or exertion undergone by the patient, which the heart is unable to answer , Dyspnoea results and it tends to be of the Inspiratory type for the reason that the negative pressure in the Thorax so produced facilitates circulation through the lungs to the greatest advantage.

The Ortho-pnoea seen so often in those advanced cases, is doubtless partly for the purpose of allowing free play to the muscles of extraordinary Inspiration .

The patient suffering from Mitral disease is apt to have exacerbations of his dyspnoea by night - he is often seen sitting propped up in bed exhausted and anxious looking, his whole chest heaving in deep rapid gasps, alae nasi and all the muscles of extraordinary respiration in full work.

When the lungs have been the seat of chronic venous congestion for a long time the capillaries in the alveolar walls become thickened and varicose. This according to Traube may also contribute towards interfering with the interchange of gases by encroaching on the alveolar space, - and also by hindering the process of diffusion.

Dyspnoea in Pericarditis.

In Pericarditis with effusion the degree of Dyspnoea depends largely on the extent of the effusion/

effusion - but pain also helps to produce it. Very large effusions not only hamper the action of the heart by the fluid pressure round it, but also interfere with the action of the diaphragm and compress the lungs even to cause collapse in some part of them. The Bronchi may also be displaced and compressed.

The character of the breathing will vary according as to one or the other of these factors predominate, generally speaking the breathing is very rapid and shallow.

In Chronic Pericarditis.

When extensive adhesions exist between the two layers of the Pericardium so that the heart is gripped and compressed, Dyspnoea may result from embarrassment of its action.

Extra Pericardial adhesions from old Mediastinitis also produce the same effect.

The right heart is said to be specially liable to sudden break down in these cases because its walls are thinner and weaker, they may be still further weakened by Myositis secondary to the Pericarditis.

Dyspnoea in Aortic Obstruction.

Patients suffering from Aortic obstruction often complain of a feeling of great oppression in the/

the chest, as if there was a heavy weight on it - without respiration being either quickened or deepened.

It is in the last stages of the disease when the auriculo ventricular valves have become incompetent and a general break up of compensation is imminent, that Dyspnoea becomes a prominent symptom.

These patients are also liable to spasmodic attacks of Angina and Dyspnoea here plays a prominent part.

It differs from the former, in that the attacks may occur at an early period in the disease, and also by the spasmodic character.

The patient usually after some fatigue or excitement, complains of sudden violent pain in the chest, he becomes pale and then livid, while the respirations become exceedingly shallow, as though the pains made him afraid to breath, and every now and again he gasps for air.

Opinions differ as to the cause - some favour Neuralgia of the Cardiac Plexus- some the theory that it is due to implications of some of the cardiac nerves in some inflammatory material or growth. But the fact that in so many cases some organic mischief is found at the root of the Aorta, justifies one in suspecting this.

The true cause is said by Gaskell to be a cramp of the cardiac muscle induced by over fatigue, complied with Ischaemia, from implications of the Coronary arteries. Which ever of these theories is/

is correct, the result is a great disturbance of the whole circulation and the Pulmonary circulation suffers with it.

In addition to the above there may be superadded a Vaso Motor spasm whereby all the arteries of the Pulmonary and Systemic circulation are constricted and the resistance offered to the Cardiac systole correspondingly increased while the Ischaemia induced in the lungs may still further add to the respiratory embarrassment.

It will only entail repetition to enter into all the cardiac disturbances which may produce Dyspnoea - since the effect produced in all is practically similar in this respect, namely engorgement of the lungs and impeded Pulmonary circulation.

But it remains to say that in the last stages this may be still further embarrassed by oedema of the lungs and fluid effusions into the Pleurae and Peritoneum.

Obstruction to the flow of blood existing in the lungs themselves.

Obstruction to the flow of blood though the lungs may be due to three causes.

1. To occulusion of the Pulmonary arteries.
2. To destruction of capillary area
3. Narrowing of the Calibre of the vessels and Capillaries.

All/

All these imply increased strain on the right heart, and it failing to overcome the resistance a reduction in the quantity of blood circulating through the lungs.

In the first and second orders, the blood has to be forced through smaller systems of vessels.

In the third the blood has to pass through vessels of diminished calibre.

Occlusion of Pulmonary Arteries.

This occurs in Embolism and Thrombosis. If one of the main branches of the Pulmonary artery is blocked, the condition is incompatible with life, the patient dies Asphyxiated almost immediately. If a medium sized branch only life may be prolonged for a time, during which the patient suffers from agonising dyspnoea = the whole body convulsed, the face livid, eyeballs starting and pupils dilated.

There may be remission with recurrent attacks, due probably to alterations in position of the Embolus. The following case illustrates this .
A woman aged forty nine, suffering from varicose ulcer in the leg, developed Phlebitis, was suddenly seized with violent pains in the chest and dyspnoea, when seen her face was livid, pulses very feeble, respiration extremely rapid and laboured.

In the course of a few minutes she improved somewhat though she remained very collapsed and was still dyspnoeic.

Later/

Later on at night she was seized again with a violent attack of dyspnoea, and coughed up some bloody sputum. she rapidly became delirious then unconscious and died.

The post mortem revealed a clot impacted in a large branch of the Pulmonary artery.

How great an obstruction may exist in the Pulmonary circulation without causing death is difficult to say.

Lichtheim found by experiments on rabbits whose Pulmonary artery he ligatured, that sufficient compensations might exist to maintain life, even when so much as three quarters of the Pulmonary circulation was put out of action - beyond this compensation failed.

Probably this power is not nearly so great in the human subject and in old or feeble persons the power of compensation would be correspondingly small.

Small emboli in the terminal twigs of the Pulmonary artery usually cause no respiratory disturbance, only a little stitch like pain and slight Haemoptysis.

Destruction of Capillary Area.

Emphysema is such an example and in this case the/

the destruction of the Capillaries in the alveolar walls may be very extensive. Only part of the lung is therefore available for circulation, and if the same amount of blood has to pass through this diminished capillary area, it follows that the obstruction so caused must raise the Intrapulmonary blood pressure and increases the strain on the right heart - which becomes hypertrophied to meet the demand and then dilated.

Then if owing to the increased strain it gives way, the congestion which may ensue still further aggravate matters.

Results of compression of the lungs on Pulmonary circulation.

Examples of such pressure is that due to gas or fluid in the Pleural cavities, one result of this is a diminution of the calibre of the vessels and capillaries in the part affected.

Even of the lung is completely collapsed circulation still goes on in it, though less easily than in mere compression. More force is required to overcome the resistance offered by the narrowed vessels.

Since such compression is apt to come on more or less suddenly the dyspnoea is correspondingly severe.

Obstruction/

Obstruction to Pulmonary circulation due to Vaso Motor Spasm.

This has already been referred to under Aneurysm and Angina pectoris.

There remains a condition known as Angina. Vaso motoria, False Angina. This consists in a general vaso motor disturbance and Cardiac spasm.

In this disease marked dyspnoea is often seen, without any organic disease of the heart.

The following case illustrates this.
The patient was a woman aged 37, she had suffered from occasional attacks of cardiac pain and dyspnoea for several years.

Sometimes they occurred two or three times a week, at other times an interval of two or three months separated the attacks.

She was a healthy looking woman, well nourished accustomed to moderately hard work, shepherds wife. She was of rather a nervous temperament. There was no history of Gout Rheumatism alcohol or syphilis.

The heart was of normal size, sounds clear , no audible murmur.

Pulse 80. regular in time and force. artery walls healthy.

Urine no albumin or other abnormal constituents.

Tongue/

Tongue rather flabby indental and furred.

Appetite poor as a rule, she suffered much from Dyspepsia unless very careful in her diet.

The attacks came on suddenly, sometimes preceeded by a tingling sensation in the extremities. A sensation as if the heart was being gripped tightly caused her to scream with pain, the breathing was very tumultuous and there was an intense desire for air. During an attack she was pale, pulse at the wrist almost imperceptible, extremities cold.

After the attack she felt exhausted for some time - and then she often passed a considerable quantity of pale urine of low specific gravity.

She was given a mixture of Ammonia and ether and took Trinitrin Tabloids which she said relieved the attacks. Attention was paid to her digestive organs and general health, and since she has improved in this respect, she has been free from attacks.

In this case I believe that the dyspnoea was to a large extent due to Vaso Motor spasm and Ischaemia of the lungs with embarrassment of the hearts action - and nervous apprehension of an attack.

Dyspnoea depending upon changes in the blood.

Whatever type of Anaemia may be present whether it depend on deficiency of Haemoglobin or red/

red corpuscles, or both, the result will be that a given volume of blood as it passes through the lungs will be able to take up less oxygen, than the same quantity of healthy blood in the same time.
Thus in order that the tissues should be properly supplied with oxygen, the blood will have to circulate more rapidly.

As a matter of fact however the anaemic patient as a general rule does not suffer from dyspnoea unless they undergo more or less exertion, because the metabolism of such patients is at a low ebb, consequently there is less need for oxygen under their ordinary circumstances. But immediately any exertion is attempted Dyspnoea ensues - the reason being that insufficient reserve of oxygen exists to meet the demand of increased consumption.

The heart too is weakened as a result of the disorder and frequently fatty - so its attempts to drive the blood more quickly through the lungs are insufficient, which the Carbon dioxide accumulating in the blood stimulates the Respiratory mechanism to increased activity.

In the anaemia of advanced Phthisis and other wasting diseases and in some cases of pernicious Anaemia and Chlorosis, Dyspnoea may not appear even after considerable exertion, for the reason that in these patients the muscular weakness is so great

and/

red corpuscles, or both, the result will be that a given volume of blood as it passes through the lungs will be able to take up less oxygen, than the same quantity of healthy blood in the same time. Thus in order that the tissues should be properly supplied with oxygen, the blood will have to circulate more rapidly.

As a matter of fact however the anaemic patient as a general rule does not suffer from dyspnoea unless they undergo more or less exertion, because the metabolism of such patients is at a low ebb, consequently there is less need for oxygen under their ordinary circumstances. But immediately any exertion is attempted Dyspnoea ensues - the reason being that insufficient reserve of oxygen exists to meet the demand of increased consumption.

The heart too is weakened as a result of the disorder and frequently fatty - so its attempts to drive the blood more quickly through the lungs are insufficient, which the Carbon dioxide accumulating in the blood stimulates the Respiratory mechanism to increased activity.

In the anaemia of advanced Phthisis and other wasting diseases and in some cases of pernicious Anaemia and Chlorosis, Dyspnoea may not appear even after considerable exertion, for the reason that in these patients the muscular weakness is so great

and/

and the irritability of the nervous centres so low, that neither react to stimuli which would otherwise produce dyspnoea.

Dyspnoea due to Toxic Causes.

Even in health toxic bodies are excreted as the result of tissue waste. It is only the nicely regulated balance between Metabolism and elimination of [illegible] products that is the safeguard against Toxaemia . Very probably in many of the diseases that have been mentioned toxic bodies circulating in the blood may assist in producing Dyspnoea.

In fever where there is increased Metabolism and frequently impaired excretory function, there is very little doubt that Dyspnoea results to a large extent from accumulation of such waste products in the blood acting upon an enfeebled and irritable Medulla.

The chief organs for excretion of waste products of course the kidneys, and it is when these are diseased that one might expect to find the effects of retention of waste products in the blood.

Uraemic Dyspnoea.

There are several types of Dyspnoeamet with in Brights disease. There is that due to the associated condition of the heart, and this may be complicated on its failure by oedema of the lung and hydrothorax. /

hydrothorax.

There are the violent respiratory efforts during the Epileptic fits of Uraemia which are merely due to the share the respiratory muscles take in the general muscular excitement.

Cheyne Stokes breathing may be present but it is that known as Uraemic Asthma which is of the greatest importance for the present discussion.

Uraemic Asthma.

This is most probably due to irritating effects produced by the retention of toxic substances in the blood, on the Vaso Motor centre in the Medulla. The general blood pressure is raised during an attack which might be expected from the respiratory embarrassment. It depends on more than this however, namely upon spasm of the Pulmonary artery and more especially of its small branches. Dickinson.

The Inspiratory character of the respiration also suggest this, and it resembles in this respect the type seen in some of the cases of impeded circulation in the lungs which have been referred to - and of which if this theory is correct it is another example.

In one case of this kind I seemed to be able to detect reduplication of the second sound during an attack, which was not present before or after - and/

and if this sign may be interpreted as a sign of increased Interpulmonary pressure it might be taken as additional evidence in favour of this theory.

Indeed as far as my experience goes I have found a reduplication of the second sound in nearly all the cases I have had the opportunity of examining during a seizure of this kind.

The attacks occur chiefly at night and come on suddenly, the patient is obliged in bad cases to sit up - his breathing is terribly distressed. Inspiratory effort is marked and exspiration laboured prolonged and hissing.
An attack may be over in a few minutes but I have seen one last over two hours.
When the attack begins to subside, then respiration gradually becomes easier and finally normal again but there remains considerable wheeziness for some time.

The attack may be brought on apparently by nervous shock.
A patient on mine, a case of large white kidney, who had never had an attack of Dyspnoea, though she had some Uraemic manifestations, suddenly had an attack, on hearing of a family bereavement, and from that time till her death soon after, very little excitement seemed to determine a seizure.

<u>Gouty Asthma.</u>/

Gouty Asthma.

Gouty subjects are notorously liable to attacks of Bronchitis and knowing the sensitiveness of the Respiratory centre to Toxic influences, I don't think it is going too far to attribute the attacks, at all events to a great extent, to irritation of the Medulla from excess of Uric acid in the blood.

Moreover in many cases there is no characteristic pathological change to be found in the lungs in Gout, so the attacks must be due to a temporary influence of some sort - but whether it is a vaso motor disturbance or due to muscular spasm of the small bronchi I am not prepared to say and this consideration together with the fact I have observed at least in one case, that the asthmatic attacks are apt to occur most frequently, just before an attack of Gout, when the blood may be assumed to contain a large amount of Uric acid - leads one to suspect that a Toxic influence may be at the root of the matter.

Dyspnoea in Diabetes.

In some cases of Diabetic coma, especially that kind which is more insidious in its onset, a characteristic Dyspnoea is seen, it is sometimes described as air hunger. The respirations are remarkably/

remarkably deep, slow and sighing.

It appears to be part of the general Toxaemia which is said by some to be due to Acetone in the blood, others including Stadelmann attribute it to β oxybutyric acid.

Lepiur denies that β oxybutyric acid is toxic to the respiratory centre at all events in dogs and cats even when given in large doses; he finds however that β Amydo Butyric acid given to dogs or cats does cause increased amplitude of respirations and also acceleration of the pulse rate and coma. This acid has not been isolated from the urine or organs of patients suffering from diabetes but it is derived from the disintegration of tissue proteids and may be an intermediate product during the formation of β oxybutyric acid, since β Amydo Butyric acid when hydrated gives rise to β oxybutyric acid and ammonia.

Grube has also by a series of experiments confirmed these results and states that the respiratory type is very similar to that observed in Diabetic Coma.

Many other poisons have been suggested at different times such as Acetone, Diacetic acid, Ammonia etc., but all have failed to satisfactorily reproduce the symptoms on experiment.

Lipaemia has alsobeen cited as a cause since the/

the capillaries in the lungs have been found in some cases to be blocked by Emboli of fatty droplets - but the extent to which this occurs is not sufficient to solve the question satisfactorily.

Moreover there is no evidence of any obstruction in the lungs either to the entrance of air or to the circulation of blood - in fact: unless the lungs happen to be the seat of some organic disease such as pneumonia or phthisis, a common enough condition in these cases; physical signs may be, indeed generally are entirely absent.

The toxic theory therefore seem the most acceptable.

It acts probably on the nervous centres in the Medulla, but exactly how it acts is difficult to say.

Dyspnoea of Nervous origin.

This part of the subject has already been encroached upon to some extent. Those lesions which have been dealt with before will only be alluded to again.

For the purpose of description I shall divide this heading into.

1. Dyspnoea resulting from functional derangement of the nervous system.
2. Dyspnoea resulting from irritation on paralysis of Peripheral nerves.

3./

3. Dyspnoea of Cerebral origin.

Dyspnoea from functional derangements of Nervous System.

Under this head might be included those cases of Pseudo Angina where there exists no organic lesion and those of Spasmodic Asthma which seem to depend chiefly upon an ill balanced, irritable state of the Vaso Motor and respiratory centres.

Another form of hysterical dyspnoea is also occasionally seen - it consists of an exaggeration of the Inspiratory act,a sort of catch in the breath, or the respiration may be very rapid 40 or more in the minute, without disturbance of pulse or distress.

2. Dyspnoea the result of irritation or paralysis of peripheral nerves.

Lesions of inferior laryngeal nerves, those have already been dealt with.

Lesions of Phrenic Nerve.

Paralysis of one Phrenic alone leads to no marked embarrassment of breathing except perhaps on exertion.

If both are involved there will be marked dyspnoea on the slighted exertion all the muscles of extraordinary Inspiration being used.

Causes may be Alcoholism Neuritis, Diphtheritic paralysis, Beriberi, Lead poisoning, Bulbar paralysis, Progressive muscular Atrophy and syringo/

syringo myelia.

The act of Inspiration is of course chiefly interfered with -the abdominal walls are then retracted since the abdominal contents are no longer pushed down by the action of the Diaphragm and the lower ribs are expanded since the contraction of Diaphragm no longer pulls them in.

The act of Expiration is said to be slowed probably on account of the imperfect expansion of the lungs and consequent insufficiency of the afferent stimuli to the respiratory centre.

The abdominal walls are distended during Expiration, and there is often orthopnoea, which position the patient assumes, presumably for the purpose of aiding the depression of the Diaphragm during inspiration by bringing the force of gravity to bear on the abdominal viscera.

Paralysis of the muscles that raise the ribs. In such cases Inspiration has to be performed by the Diaphragm alone.

The chest walls remain immoveable in the position of Expiration. Less air is taken in at each inspiration so the number of respirations has to be increased.

The respirations therefore are shallow, rapid and abdominal in type.

Life/

Life may be maintained in this condition for a considerable time, for example T.B. labourer aged 35 was admitted into the Infirmary with fracture dislocation of the spine about the upper Cervical region _the trunk, and both upper and lower extremities were completely paralysed, the Diaphragm was alone available for respiration, which it managed successfully during the four months he was in Hospital. His breathing was shallow and rapid and abdominal, but without distress unless he was moved.

He had great difficulty in getting rid of Bronchial secretions by coughing, power was so weak, but he never had any serious Pulmonary complications.

Laryngeal crisis in Locomotor Ataxy.

The severe laryngeal symptoms occasionally manifested in this disease; consisting of laryngeal spasm, with stridor and Inspiratory dyspnoea are due in all probability to a transient disturbance of the nervous centres controlling the recurrent laryngeals. The symptoms are those of laryngeal obstruction as described elsewhere.

Effect of Cerebral lesions on Respiration.

Horsley and Spencer have shewn by experiments on animals that one effect of Intra cranial pressure is/

is to produce slowing and deepening of respiration.

It is supposed that Anaemia of the Medulla is produced whereby it becomes more irritable and the action of the vital centres more unstable.

Thus the heart is slowed and the respiration deepened from excitement of the Cardio Inhibitory and respiratory centres respectively.

Later, when the centres become exhausted both pulse and respiration become rapid and irregular.

The above events are commonly seen in the human subject in cases of Meningeal haemorrhage, haemorrhage into the substance of the Brain and ventricles. In Meningitis and in all cases where the Intracranial pressure is increased somewhat rapidly.

Rosenthal has observed in cases of Meningitis that the condition of the pulse and respiration correspond with the extent and intensity of the exudatative process.

In the earlier stages of compression respiration is slow and deep - but when compression becomes very great they may be increased to thirty or forty a minute or more. Other observers have noticed a tendancy towards periodicity in the early stages like Cheyne Stokes breathing but differing from it in that the depth of the respiration following one another/

another do not vary, but three or four equally deep respirations are taken, then a pause of a few seconds complete Apnoea ; and so on.

In the later stages true Cheyne Stokes breathing is often seen.

The question whether these effects are due directly to Compression and Anaemia of the Brain is I think perhaps debatable, undoubtedly compression does produce anaemia, but it also produces cerebral paralysis, and having regard to the fact taught us by Physiology that the result of cutting off the paths of Cerebral influences from the respiratory centre in the Medulla is to deepen and slow respiration, it would seem as though suppression of these influences from Cerebral paralysis independently of Anaemia due to compression, might have at all events some voice in the matter.

Take the case of apoplexy, unless the effusion is considerable, it would hardly be capable of producing any degree of compression sufficient to produce anaemia. and yet I think I may be allowed to say sometimes even comparatively small haemorrhage may cause insensibility if very sudden in onset; from shock to the brain - the higher centres are then paralysed, it may be only a temporary paralysis, but till the brain recovers; its controlling influences/

influence on the Medullary centre is in abeyance with the result of deepening and slowing respiration.

Also in sleep, when the cerebral functions may be said to be temporarily suspended the respirations are generally slow and deep, and may also exhibit periodicity, here there is no question of increased intracranial pressure, so unless one attributes it entirely to Cerebral Anaemia, it seems reasonable to suppose that it may be at all events partly due to diminished cerebral control.